Bottom Line on Diabetes and Insulin Resistance

Insulin resistance = cannot make new fat.

Eat: ⇑ Insulin from the pancreas.
Glucose (→ Glycogen) is stored in the liver.
Glucose (via De Novo Lipogenesis) → fat.

Type I – No insulin = glucose cannot get into the liver & be made into fat = skinny.
Type II – Too much sugar (Fructose & Glucose).
The liver gets loaded with too much sugar + fat.
No more room for either = "Fatty Liver."

THE KEY TO UNDERSTANDING INSULIN RESISTANCE IS "FATTY LIVER."
IT'S AN "OVERFLOW ISSUE" – SUGAR CANNOT GET INTO THE LIVER.
*Note: Eating fat does not make you fat. It is digested like this:

1) Bile breaks it down.
2) Packed into Chylomicrons.
3) Into the lymph system.
4) Then to the bloodstream.
5) BOTTOM LINE: FAT IS NOT METABOLIZED IN THE LIVER.

THEREFORE, IT'S THE GLUCOSE/FRUCTOSE THAT LEADS TO FAT STORAGE.

- FATTY CHO = ⇑ INSULIN OVER TIME.
- FATTY LIVER (PACKED FULL VIA DE NOVO LIPOGENESIS).

- = INSULIN RESISTANCE AND THE SUGAR THEN GOES ALL OVER THE BODY AND CREATES HAVOC.
- FRUCTOSE DOES NOT INCREASE INSULIN.
- IT IS ONLY METABOLIZED IN THE LIVER.
- SUGAR = ½ GLUCOSE + ½ FRUCTOSE.
- THE ½ GLUCOSE IS METABOLIZED AND SENT TO ALL BODY TISSUES.
- A 170 LB. PERSON = ALL GLUCOSE CAN BE METABOLIZED BY 170 LBS. OF TISSUE (BUT EXCESS CAN COMPOUND WITH EXCESS FRUCTOSE TO CREATE OVER-FILL).
- THE ½ FRUCTOSE IS METABOLIZED BY A FIVE-LB. LIVER.
- BUT IF THE LIVER IS FATTY & GLUCOSE STORES ARE COMPLETELY FULL ALL OVER THE BODY, THE LIVER TURNS THE FRUCTOSE INTO FAT (AS MUCH AS IT CAN, CONTINGENT ON THE FUNCTION OF THE OVER-FILLED LIVER).
- STARCHES/POLYS/COMPLEX – ALL GLUCOSE & METABOLIZED BY THE ENTIRE BODY.
- FRUCTOSE/MONOS/SIMPLE – LIVER-ONLY METABOLIZED & TURNED INTO FAT (DE NOVO) BUT CANNOT BE PROCESSED BY A FATTY LIVER = INSULIN RESISTANCE.

<u>TOO MUCH SUGAR FROM ABOVE SITUATIONS</u>:

- In the entire body, not just the bloodstream.
- Blood sugar levels do not clearly indicate whether you're healthy or not.

REVERSING TYPE II DIABETES

1) Decrease sugar over time.
   a) ↓ liver fat.
   b) ↓ pancreas fat.
2) Consume a low CHO diet.
3) Burn off excess fat via intermittent fasting.

Medications:

Acarbose blocks the absorption of sugar, but not blood sugar. It has been shown to reduce the risk of heart attack by 50%.

Empa-Reg – pee out sugar but does not lower blood glucose. Also, it has been shown to reduce the risk of heart attack by 50%.

Treating type II diabetes:

- It's a disease of insulin resistance, NOT too much blood sugar.
- Don't treat the symptoms (high blood sugar), but treat insulin resistance because of too much insulin:
  A) Fatty liver/overflow issue.
  B) Too much insulin due to that fatty liver.

UPSIDE OF INTERMITTENT FASTING

- Protein & fat do increase insulin levels, and if you have high levels of it, fasting can lower any level of insulin.
- Fasting = no food = zero insulin = more glucagon = energy from stored fat.

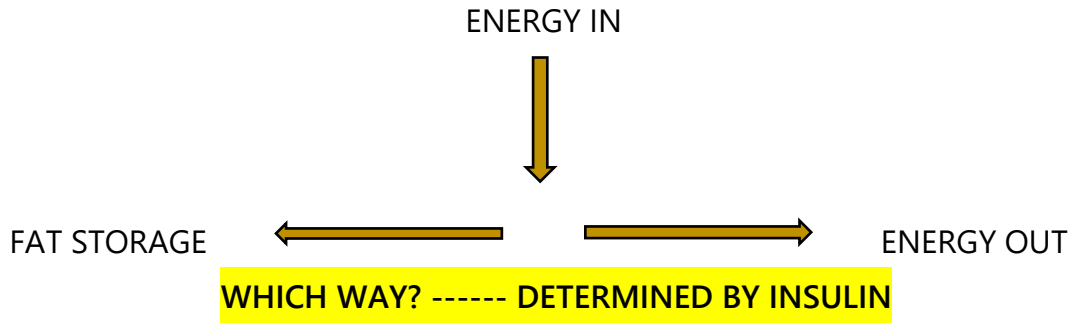
PROBLEM WITH CALORIES IN VS. OUT

↓ Calories = ↓ BMR

Also decreases in heart volume, rate, and body temperature.

Insulin and, somewhat, Cortisol ↑ = fat storage.

Obesity weight gain is a 2-compartment problem:



Problem with long-term calorie decrease:

- Hungrier
- Hormones make you hungrier.
- BMR does decrease, but no net calorie difference.

CRUEL HOAX

EAT LESS

INCREASE HUNGER

DECREASE METABOLISM

HORMONES MAKE YOU REGAIN WEIGHT

Proximal vs. Ultimate Goals:

ULTIMATE GOAL	PROXIMAL GOAL	RESULT

Insulin, too many calories, obesity

POPULAR THOUGHT, BUT WRONG:
No discipline/gluttony / personal choice → Too many calories = Obesity

SCIENCE-BASED HORMONAL OBESITY - THE ACTUAL TRUTH:
High Insulin --------- Obesity ---------- too many calories

What can you do?

1. Decrease simple sugars, which increase insulin.
2. Vinegar?
3. Increase high-fiber CHO.
4. Moderate protein intake = moderate insulin increase.
5. High natural fat intake = lower insulin rise.